Intensive Care Med
https://doi.org/10.1007/s00134-025-07782-8

EDITORIAL

How we could use critical care echocardiography in the assessment of and management of cardiovascular phenotypes in septic shock: the good, the bad, and the ugly profiles

Antonio Messina[1,2*] and Antoine Vieillard-Baron[3,4]

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Septic shock is a life-threatening time-dependent medical emergency that still kills 30–40% of patients. It results from a dysregulated host response to infection, leading to acute circulatory failure and organ dysfunction [1, 2]. Clinical manifestations of septic shock are highly variable, and they are influenced by factors like age, underlying health condition, and infection source [3]. As a result, conventional generalized treatments have been increasingly challenged in favor of personalized approaches tailored to the individual patient's response [4, 5].

Personalization aims at optimizing initial therapy, by combining evidence-based guidelines with customized care [6]. Critical care ultrasonography [CCUS, including critical care echocardiography (CCE)] plays a pivotal role in this context, being an adaptable and widely available component of the evaluation and tailored clinical management of patients with septic shock [7]. Moreover, CCE has facilitated the classification of shock subtypes based on hemodynamic parameters estimated noninvasively [8], and has modified the classical evolution of hemodynamic status in septic shock from "static" phases to an adaptative heart vessels interplay, also demonstrating that cardiac failure may occur very early [9]. Even though basic CCE skills enable a prompt recognition of cardiovascular phenotypes, including sepsis-induced left ventricular (LV) systolic dysfunction, more advanced measurements can guide the titration of the initial therapy. Depending on the definition used and the timing of CCE assessment, the prevalence of sepsis-induced LV systolic dysfunction may vary between 10 and 70%, and some pre-existing condition may be overlapped with an acute dysfunction [10].

In this article, we propose a classification of different cardiovascular phenotypes potentially observed in septic shock into three categories/profiles, promptly recognizable by CCE, according to their potential respective impact on the outcome and the adaptation of treatment (Fig. 1). While it will not be discussed here, it is also well-documented that the right ventricle may also be affected by heart–lung and interventricular interactions, as well as by inflammation, and that its dysfunction is associated with a poor outcome [11].

Clinical classification is complex because cardiovascular profiles are dynamic and patients may move from one to another according to fluid administration, correction of LV afterload, and evolution of the disease. Therefore, CCE must be repeated, as explained below.

1. The "*good*": This profile is characterized by normal systolic function, with preserved LV ejection fraction (EF), normal ventricular size and absence of wall motion abnormalities. While these patients may be considered as well resuscitated, they may still be in shock and their mortality has been reported around 21% in one paper [8]. They may sometimes

*Correspondence: antonio.messina@humanitas.it
[1] IRCCS Humanitas Research Hospital, Rozzano, Milan, Italy
Full author information is available at the end of the article

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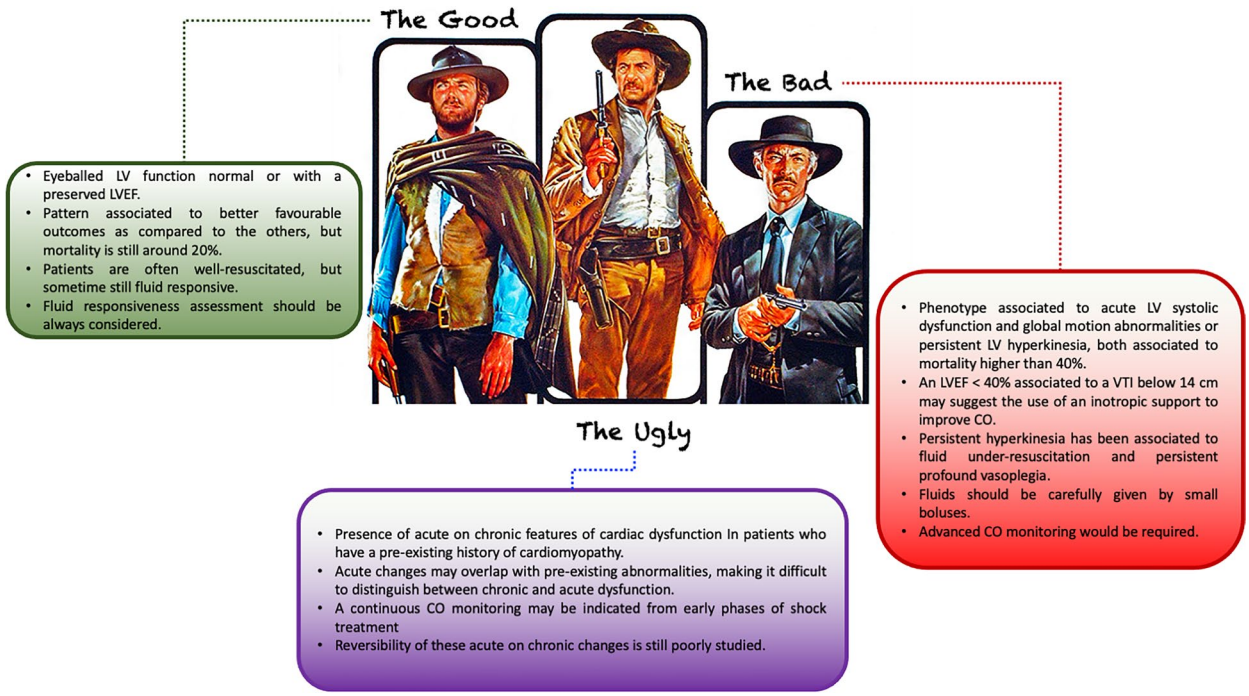
The Good
• Eyeballed LV function normal or with a preserved LVEF.
• Pattern associated to better favourable outcomes as compared to the others, but mortality is still around 20%.
• Patients are often well-resuscitated, but sometime still fluid responsive.
• Fluid responsiveness assessment should be always considered.
The Bad
• Phenotype associated to acute LV systolic dysfunction and global motion abnormalities or persistent LV hyperkinesia, both associated to mortality higher than 40%.
• An LVEF < 40% associated to a VTI below 14 cm may suggest the use of an inotropic support to improve CO.
• Persistent hyperkinesia has been associated to fluid under-resuscitation and persistent profound vasoplegia.
• Fluids should be carefully given by small boluses.
• Advanced CO monitoring would be required.
The Ugly
• Presence of acute on chronic features of cardiac dysfunction In patients who have a pre-existing history of cardiomyopathy.
• Acute changes may overlap with pre-existing abnormalities, making it difficult to distinguish between chronic and acute dysfunction.
• A continuous CO monitoring may be indicated from early phases of shock treatment
• Reversibility of these acute on chronic changes is still poorly studied.

Fig. 1 Schematic features of hemodynamic profiles. *LV* left ventricle, *EF* ejection fraction, *VTI* velocity time integral, *CO* cardiac output

still be fluid-responsive, even though fluid therapy, if decided, must be titrated very carefully after initial fluid resuscitation, using small fluid boluses and assessing their effect on tissue perfusion [12]. For that purpose, CCE may be of major help in managing the need for fluids [13], including the evaluation of pulmonary and systemic signs of edema and venous congestion [14], while more data are still needed to clarify its exact value to assess the balance between fluid responsiveness and fluid tolerance.

2. The *"bad":* This profile is characterized by either an acute and severe LV systolic dysfunction or a LV hyperkinesia, both associated with higher in-hospital mortality (51 and 41%, respectively) in one large retrospective cohort of patients with sepsis and septic shock [15]. LVEF does not reflect intrinsic myocardial contraction but the coupling with LV afterload. Then LV hyperkinesia may reflect a persistent and profound vasoplegia, despite norepinephrine infusion. In this case, even if an LV end-systolic cavity obliteration is observed (i.e., the "kissing" LV)", LV end-diastolic size is still normal and CCE suggests to stop fluid administration. In this subgroup of patients with tachycardia, the clinical benefit of beta-blockers is still questionable [16], and ongoing trials, such as the HyperBetaschock (NCT 04748796), will provide information on this topic.

However, there is another situation where LV is hyperkinetic, but the end-diastolic size is small and CCE suggests to give more fluids, which is fluid under-resuscitation. It has been reported that this profile, when a coexisting dynamic obstruction is present, is associated with a high mortality rate of approximately 55% [17].

Severe LV systolic dysfunction may result either from profound depression of intrinsic contractility, or to over-correction of blood pressure. Besides an extreme situation with an LVEF < 25%, representing around 4% of the cohort in the Dugar's paper [15], how sepsis-induced LV systolic dysfunction may be accurately defined and its association with a poor outcome is still questionable. Geri et al. suggested that a LVEF < 40% associated with an aortic velocity time integral (VTI) below 14 cm could be an adequate definition. It makes sense as it associates low cardiac output related to LV systolic function depression, in patients resuscitated with fluids and vasopressors, but with still high lactate level [8]. Some studies reported that, in this case, dobutamine infusion could correct cardiac function, restore cardiac output, and decrease heart rate [18].

However, it is still unknown whether this approach could improve outcomes. It was indeed also suggested that LV systolic dysfunction could only reflect myocardial hibernation, which is an adaptative, self-regulated, and reversible metabolic and functional response to infection [19]. Again, ongoing randomized controlled trials, such as the ADAPT (NCT 04166331), will bring information in the next few years.

3. The *"ugly"*: This profile is observed in patients with septic shock who have a pre-existing history of LV dysfunction due to conditions such as ischemic heart disease, diabetes, chronic hypertension, or renal failure. In these cases, the echocardiographic pattern can be particularly challenging to interpret. These patients often exhibit chronic cardiac abnormalities, such as left ventricular hypertrophy, associated or not with LV dynamic obstruction, dilated ventricles, or impaired systolic and diastolic function. In addition, valvular diseases, including mitral regurgitation or aortic stenosis, may further complicate the cardiac assessment. In patients with chronic cardiac conditions, these acute changes may overlap with pre-existing abnormalities, making it difficult to distinguish between chronic and acute dysfunction. As an illustration, a patient with chronic ischemic heart disease may already have regions of myocardial akinesis or dyskinesis, complicating the assessment of new septic-induced dysfunction. Similarly, chronic hypertrophy due to hypertension or diabetic cardiomyopathy may mask the extent of septic-related left ventricular dysfunction. Moreover, the reversibility of these acute changes is still poorly studied. Therefore, an early CCE assessment upon admission is crucial to establish a baseline examination for comparing potential new LV impairments during the treatment.

In conclusion, cardiac dysfunction is common in patients with sepsis and septic shock; however, its presentation may significantly vary in response to pathophysiological variables (i.e., preload, afterload and heart rate) and pre-existing heart dysfunction [11]. It leads to different cardiovascular profiles with potentially different outcomes and management that may be assisted and titrated by CCUS, together with clinical and hemodynamic findings. Specifically, checking fluid responsiveness and tolerance, together with the systemic response to vasopressors and inotropes are key purposes of CCUS, driving physicians toward the most appropriate clinical decisions at the bedside. CCE has to be repeated to personalize therapy, because of possible transitions among different phenotypes.

Author details
[1] IRCCS Humanitas Research Hospital, Rozzano, Milan, Italy. [2] Department of Biomedical Sciences, Humanitas University, Milan, Italy. [3] Intensive Care Medicine Unit, Assistance Publique-Hôpitaux de Paris, University Hospital Ambroise Pare, Billancourt, Boulogne, France. [4] INSERM UMR-1018, CESP, Team Kidney and Heart, University of Versailles Saint-Quentin en Yvelines, Villejuif, France.

Conflicts of interest
The authors have no conflicts of interest to be declared for the present paper.

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Received: 16 October 2024 Accepted: 31 December 2024
Published online: 20 January 2025

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